Pancreas divisum: Clinical manifestations and diagnosis

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INTRODUCTION

Congenital anomalies and variants of the pancreas are seen in approximately 10 percent of the general population. While many are found coincidentally, a portion of these anomalies and variants are clinically significant and cause symptoms in childhood or adulthood.

This topic will review the epidemiology, pathogenesis, clinical manifestations, and diagnosis of pancreas divisum. The management of pancreas divisum is discussed separately. (See "Treatment of pancreas divisum".)

EPIDEMIOLOGY

Pancreas divisum is the most common congenital pancreatic anomaly, occurring in approximately 10 percent of individuals [1-3]. The frequency with which pancreas divisum is seen on endoscopic retrograde cholangiopancreatography (ERCP) depends upon the number of patients with pancreatitis in the study population and the completeness of pancreatography. In one retrospective study of 1825 successful consecutive ERCPs, pancreas divisum was found in 7.5 percent [4]. The prevalence of pancreas divisum was significantly higher in patients presenting with idiopathic pancreatitis as compared with controls (50 versus 4 percent). In another retrospective study that included 809 patients who underwent ERCP, pancreas divisum was more frequent in patients with pancreatitis as compared with those with biliary diseases or obscure abdominal pain (9 versus 2 percent) [5].

EMBRYOLOGY

Normal pancreatic duct development — The pancreas is formed from fusion of the dorsal and ventral anlages, which develop from the embryologic foregut (figure 1) [6]. The ventral system also gives rise to the hepatobiliary system. At approximately the seventh intrauterine week of life, the ventral pancreas rotates posterior to the duodenum and comes to rest posterior and inferior to the head portion of the dorsal pancreas. The ventral bud forms the inferior part of the head of the pancreas and the uncinate process, whereas the dorsal bud becomes the tail and the body. The ventral pancreas represents 2 to 20 percent of the pancreatic parenchymal mass. Fusion of the ductular network of the two buds gives rise to the main pancreatic duct. The accessory pancreatic duct (of Santorini), which often persists, is derived from the dorsal pancreatic duct proximal to the site of fusion. Fusion of the ductal system occurs in over 90 percent of individuals, although variations in patency of the accessory duct occur.

Pancreas divisum — Failure of fusion of the ventral and dorsal duct system results in pancreas divisum.

Subtypes — There are three subtypes of pancreas divisum (figure 1):

- **Classic (complete) pancreas divisum** – The classic pancreas divisum anatomy consists of a small ventral duct, which drains through the larger major papilla, and the larger dorsal duct, which drains through the smaller minor papilla. In some cases, the entire pancreatic ductal system drains through the minor papilla via the dorsal duct. Based on autopsy data, approximately 70 percent of patients with pancreas divisum have the classic form.

- **Incomplete pancreas divisum** – In individuals with incomplete pancreas divisum, a small branch of the ventral duct communicates with the dorsal duct. Approximately 15 percent of cases of pancreas divisum are of the incomplete type [7-9]. However, the clinical implications of incomplete pancreas divisum are the same as for classic (complete) pancreas divisum, except that modest to full visualization of the dorsal duct may occur with vigorous major papillary contrast injection on endoscopic retrograde cholangiopancreatogram. (See 'Endoscopic retrograde cholangiopancreatogram' below.)

- **Reverse divisum** – Reverse divisum can occur in the setting of an isolated small segment of dorsal pancreas. In reverse divisum, the accessory duct of Santorini does

not connect with the genu of the main pancreatic duct and can therefore be mistaken for a mimic of malignant obstruction. The physiologic significance of this anomaly is that the overflow valve to the main ductal system is absent. Thus, a gallstone that is impacted at the major papilla will likely cause more severe pancreatitis as compared with most cases of pancreatitis caused by pancreas divisum. (See 'Clinical manifestations' below.)

Pathogenesis of pancreatitis in pancreas divisum — As most patients with pancreas divisum are asymptomatic, there is considerable controversy as to whether pancreas divisum is associated with pancreatitis [10-15]. However, there appears to be a subgroup of patients with pancreas divisum that is subject to recurrent bouts of seemingly idiopathic pancreatitis. In these patients, the minor papilla orifice is so small that excessively high intrapancreatic dorsal ductal pressure occurs during active secretion, which may result in inadequate drainage, ductal distension, pain, and in some cases, pancreatitis [4,16-19]. It has also been suggested that even low-grade intraductal hypertension makes the pancreas more prone to injury from alcohol, trauma, and drugs [20]. As the causative lesion is hypothesized to be relative stenosis of the minor papilla rather than pancreas divisum per se, this condition has been termed the dominant dorsal duct syndrome [21,22]. Genetic studies have also suggested that many patients with pancreas divisum who have pancreatitis carry at least one allele of the cystic fibrosis gene product, suggesting a multifactorial origin of pancreatitis in such patients [23,24]. (See "Etiology of acute pancreatitis", section on 'Genetic risk' and "Cystic fibrosis: Overview of gastrointestinal disease", section on 'Pancreatic disease'.)

ASSOCIATED PANCREATOBILIARY ABNORMALITIES

Pancreas divisum can be associated with other pancreatobiliary abnormalities:

- Approximately one-third to one-half of patients with annular pancreas also have pancreas divisum, a setting in which rotational and fusional abnormalities coexist [25-29]. (See "Annular pancreas".)
- Up to one-half of patients with pancreas divisum have elevated sphincter of Oddi dysfunction [21,30,31].
- Pancreas divisum may be associated with partial agenesis of the dorsal pancreas [32,33].

CLINICAL MANIFESTATIONS

More than 95 percent of patients with pancreas divisum are asymptomatic. It is estimated that fewer than 5 percent of patients with pancreas divisum are symptomatic. Most symptomatic patients have infrequent bouts of pancreatobiliary-type pain or develop mild acute pancreatitis. However, a subset of symptomatic patients with pancreas divisum have recurrent pancreatobiliary-type pain, severe acute pancreatitis, chronic pancreatitis associated with clinically significant disability due to chronic abdominal pain or pancreatic insufficiency, or pancreatitis-associated complications (eg, pseudocysts) [34-39]. (See "Clinical manifestations and diagnosis of acute pancreatitis", section on 'Natural history and complications' and "Chronic pancreatitis: Clinical manifestations and diagnosis in adults" and "Overview of the complications of chronic pancreatitis".)

As most patients with pancreas divisum are asymptomatic, there is considerable controversy as to whether pancreas divisum is the cause of pancreatitis [10-15,24]. (See 'Pathogenesis of pancreatitis in pancreas divisum' above.)

DIAGNOSIS

The diagnosis of pancreas divisum is established by abdominal imaging. It is often diagnosed incidentally on abdominal imaging (computed tomography [CT] or magnetic resonance cholangiopancreatogram [MRCP]) in patients who are asymptomatic or have unrelated symptoms. However, pancreas divisum should be suspected in patients with idiopathic recurrent acute pancreatitis. In symptomatic patients, the imaging modality of choice is MRCP, which demonstrates the failure of fusion of the ventral and dorsal duct system. (See "Etiology of acute pancreatitis", section on 'Approach to establishing the underlying etiology'.)

Diagnostic evaluation — Our approach to the diagnostic evaluation of pancreas divisum depends on the clinical presentation.

Asymptomatic — More than 95 percent of patients with pancreas divisum do not have associated symptoms. They are found to have evidence of pancreas divisum on cross-sectional abdominal imaging performed for unrelated symptoms. In asymptomatic patients with evidence of pancreas divisum and no evidence of acute or chronic pancreatitis or its complications (eg, pseudocysts), we do not pursue further pancreatic evaluation.

CT scan usually shows nonspecific prominence of the pancreatic head and has a low sensitivity in the diagnosis of pancreas divisum when the pancreatic duct is not visualized [40,41]. If the pancreatic duct is visualized on abdominal CT scan, axial images in patients with pancreas divisum demonstrate the dorsal duct passing anteriorly and superiorly to the terminal common bile duct and emptying separately into minor papilla [42]. Dilation of the dorsal duct and/or changes of chronic pancreatitis that are confined to the dorsal area of the pancreas are suggestive of a pathologic minor papillary narrowing in patients with pancreas divisum. Visualization of a fat plane between the dorsal and ventral portions on CT scan is also suggestive of pancreas divisum [43].

Symptomatic or evidence of pancreatitis/complications — Symptoms of pancreas divisum include either infrequent bouts of pancreatobiliary-type pain or mild acute pancreatitis, or in a subset of patients, recurrent pancreatobiliary-type pain, severe acute pancreatitis, and chronic pancreatitis. In patients with pancreaticobiliary-type pain or acute or chronic pancreatitis, we perform a secretin-enhanced MRCP (S-MRCP). If needed, we also perform an endoscopic ultrasound (EUS) for a more detailed pancreatic evaluation. EUS may offer additional insight in false or "pseudo-divisum" of the pancreas. A mimic of complete pancreas divisum on MRI or endoscopic retrograde pancreatography is caused by pathologic obstruction of the ventral duct (of Wirsung) either focally or in a contiguous fashion up to its convergence with the dorsal duct of Santorini. Common etiologies include a ventral pancreatic duct stricture (benign or malignant), obstructing stones, focal pancreatitis, and/or necrosis in the ventral anlage/head of the pancreas.

For those patients without symptoms associated with pancreas divisum but evidence of pancreatitis or its complications on CT scan, we also perform secretin-enhanced MRCP. We reserve endoscopic retrograde cholangiopancreatography (ERCP) for patients with MRCP evidence of pancreas divisum in whom endoscopic therapy is planned. (See "Treatment of pancreas divisum", section on 'Approach to management' and "Endoscopic ultrasound in chronic pancreatitis".)

Magnetic resonance cholangiopancreatography — Secretin-enhanced MRCP is the imaging modality of choice for the diagnosis of pancreas divisum [44,45]. MRCP findings that are diagnostic of pancreas divisum include visualization of the dorsal pancreatic duct crossing anterior to the common bile duct and draining superiorly into the minor papilla and visualization of a separate ventral pancreatic duct [46]. Secretin enhancement has been shown to improve visualization of pancreatic ductal anatomy on MRCP. Secretin-enhanced MRCP has been reported to have a sensitivity and specificity of 83 and 99 percent, respectively, but the sensitivity is lower (73 percent) in the setting of chronic pancreatitis

[47,48].

Endoscopic retrograde cholangiopancreatogram — Diagnostic findings of pancreas divisum on ERCP with dorsal ductography include the presence of a short and thin ventral duct at the major papilla and filling of the dorsal duct at the minor papilla, draining the pancreas from the tail to the head. The presence of dorsal duct dilation and/or chronic pancreatitis of the dorsal pancreas are suggestive of minor papillary narrowing. Other findings that are suggestive, but not diagnostic, of pancreas divisum on ERCP include cystic dilation of the terminal pancreatic duct (santorinicele), slow drainage of contrast from the dorsal duct (>12 minutes), and pain provocation during dorsal ductography. In approximately 2 percent of patients, dorsal ductography shows an isolated small ductal system (isolated dorsal segment), which can simulate a typical ventral pancreas. Once detected, the major papilla must be cannulated to view the main pancreatic duct.

- **Minor papilla cannulation/dorsal ductography technique** – The minor papilla is nearly always located in the right upper quadrant of the visual field when facing the major papilla. It may be as close as 10 mm to the major papilla, or more often, as far as 2 to 3 cm (cephalad and anterior) away from the major papilla. Cannulation is generally best achieved with the endoscope pushed into the long position along the greater curve of the stomach. The tapered 5-Fr catheter with a 23- or 25-gauge blunt needle tip protruding 1 to 2 mm beyond the catheter tip is most helpful in achieving cannulation when diagnostic pancreatography is being performed [49,50]. If deep cannulation (with subsequent therapy) is needed, we typically use a 0.018- or 0.021-inch guidewire through a highly tapered 5-4-3 Fr catheter. The remainder of the procedure, including contrast media and fluoroscopic techniques, is similar to conventional ERCP. In experienced hands, minor papilla cannulation can be achieved in approximately 90 to 95 percent of cases [7,14]. Failures are usually associated with minor papilla distortion from inflammation (eg, pancreatitis or peptic ulcer disease), diverticula, or tumor.

  In patients in whom the minor papilla orifice is not evident, we administer intravenous secretin (16 mcg) to increase pancreatic exocrine juice flow and cause visible dilation of the minor papillary orifice [51]. However, administration of secretin is not without risk during pancreatography. In the presence of vigorous juice flow, it may be difficult to force contrast media retrograde into the pancreatic tail, possibly predisposing to pancreatitis. As a result, the administration of secretin during ERCP should be limited to difficult cases. If secretin stimulation does not produce a visible increase in pancreatic juice flow, the orifice may become apparent after spraying a diluted (1:10) methylene blue solution over the face of the minor papilla; clear juice will wash away the

background blue dye at the orifice [52].

ADDITIONAL TESTS FOR PATHOLOGIC PAPILLARY NARROWING

A number of tests have been used in patients with pancreas divisum to determine if they have pathologic narrowing of the minor papilla that may be predisposing them to develop pancreatitis. However, the sensitivity and specificity of these tests are largely undefined and correlation with a response to therapy has not been consistently demonstrated. We therefore do not routinely perform these tests. (See 'Pathogenesis of pancreatitis in pancreas divisum' above.)

Pancreatic juice collection — In patients who fail to respond to minor papilla therapy, bicarbonate concentration in pancreatic juice may be used to assess pancreatic duct cell function and diagnose exocrine pancreatic insufficiency, suggestive of chronic pancreatitis. Collection of less than 3 mL/min of secretin-stimulated pancreatic juice from the minor papilla with a bicarbonate concentration of less than 105 mEq/L is suggestive of chronic pancreatitis. Although data supporting such an approach have involved sampling of pancreatic juice from the major papilla, we believe that they can be extrapolated to the minor papilla. The evaluation of pancreatic exocrine function and diagnosis of chronic pancreatitis are discussed separately. (See "Exocrine pancreatic insufficiency" and "Chronic pancreatitis: Clinical manifestations and diagnosis in adults".)

Secretin ultrasound — The normal pancreas shows dilation of the main pancreatic duct over a 5- to 10-minute interval after intravenous secretin stimulation [53]. While some studies have suggested that patients with pancreatic outlet obstruction may have dorsal duct dilation, which persists for more than 15 minutes after secretin administration, this has not been consistently demonstrated [22,53-55]. The use of secretin ultrasonography may also be problematic in patients with chronic pancreatitis. Such patients may have hyposecretory exocrine function and may not manifest dorsal duct dilation even in the presence of significant minor papilla narrowing. In addition, obesity or overlying gas can preclude visualization on ultrasonography. Although one study suggested that a positive ultrasound-secretin test is a predictor of successful accessory papilla sphincteroplasty, these results have not been validated [22,55].

Diagnostic stenting — Stenting of the minor papilla to determine if ductal obstruction is the cause of pancreatitis or pain is of uncertain benefit, particularly in patients with infrequent episodes of pain (one or two times a year).

Papillary manometry and patency assessment — Manometry of the minor papilla has not been systematically evaluated, and normal minor papilla basal pressures have not been defined. As a basal pressure of >40 mmHg is considered abnormal for the major papilla, it has been suggested as a threshold for the minor papilla basal pressure. Our own limited experience with use of a standard 5-Fr triple-lumen manometry catheter (over a 0.018-inch guidewire) in previously untreated minor papillae has invariably shown very high basal pressures of >200 mmHg in patients with pancreas divisum. Prior studies have found high intraductal basal pressures in the dorsal duct of patients with pancreas divisum as compared with accessory duct pressures (minor papilla cannulation) in non-divisum patients with patent major papilla orifices [56]. However, these studies were not performed with small-caliber catheters and both intraductal and intrapapillary (intra-sphincter) pressures were not measured.

The degree of resistance to passage of a 3-, 4-, or 5-Fr catheter following passage of a guidewire into the dorsal duct has been used to gauge the degree of minor papilla narrowing. This is similar to an intraoperative assessment used by surgeons to evaluate sphincter patency; however, it has not been standardized.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of pancreas divisum includes other congenital anomalies (annular pancreas), false or pseudo-divisum, and variants of the pancreas that are seen in approximately 10 percent of the general population. Pancreas divisum can be differentiated from other ductal abnormalities by imaging the pancreatic duct with magnetic resonance cholangiopancreatography or endoscopic retrograde cholangiopancreatography (table 1). The differential diagnosis for acute pancreatitis is discussed in detail, separately. (See "Etiology of acute pancreatitis".)

SUMMARY

- Pancreas divisum is the most common congenital pancreatic anomaly, occurring in approximately 10 percent of individuals. (See 'Epidemiology' above.)
- The pancreas is formed from fusion of the dorsal and ventral anlages, which develop from the embryologic foregut. Fusion of the ductal system occurs in just over 90 percent of individuals, although variations in patency of the accessory duct (of

Santorini) occur. Failure of fusion of the ventral and dorsal duct system results in pancreas divisum (figure 1). (See 'Pancreas divisum' above.)

- More than 95 percent of patients with pancreas divisum are asymptomatic. Fewer than 5 percent of patients with pancreas divisum are symptomatic. Most symptomatic patients have infrequent bouts of pancreatobiliary-type pain or develop mild acute pancreatitis. However, a subset of symptomatic patients with pancreas divisum have recurrent pancreatobiliary-type pain, severe acute pancreatitis, chronic pancreatitis associated with clinically significant disability due to chronic abdominal pain or pancreatic insufficiency, or pancreatitis-associated complications (eg, pseudocysts). (See 'Clinical manifestations' above.)

- As most patients with pancreas divisum are asymptomatic, there is considerable controversy as to whether pancreas divisum is associated with pancreatitis. However, in a subgroup of patients with pancreas divisum with recurrent bouts of seemingly idiopathic pancreatitis, there may be pathologic narrowing of the minor papillary orifice. This results in excessively high intrapancreatic dorsal ductal pressure during active secretion, which may result in inadequate drainage, ductal distension, pain, and in some cases, pancreatitis. It has also been suggested that even low-grade intraductal hypertension makes the pancreas more prone to injury from alcohol, trauma, and drugs. (See 'Pathogenesis of pancreatitis in pancreas divisum' above.)

- The diagnosis of pancreas divisum is established by abdominal imaging. It is often diagnosed incidentally on abdominal imaging (computed tomography [CT] or magnetic resonance cholangiopancreatogram [MRCP]) in patients who are asymptomatic or have unrelated symptoms. However, pancreas divisum should be suspected in patients with idiopathic recurrent acute pancreatitis. In symptomatic patients, the imaging modality of choice is secretin-enhanced MRCP, which demonstrates the failure of fusion of the ventral and dorsal duct system. (See 'Diagnosis' above.)

- Our approach to the diagnostic evaluation of pancreas divisum depends on the clinical presentation. In asymptomatic patients with evidence of pancreas divisum on abdominal CT scan and no evidence of acute or chronic pancreatitis or its complications (eg, pseudocyst), we do not pursue further evaluation. In patients with pancreaticobiliary-type pain or acute or chronic pancreatitis, we perform a secretin-enhanced MRCP. For those patients without symptoms associated with pancreas divisum but evidence of pancreatitis or its complications on CT scan, we also perform secretin-enhanced MRCP. We reserve endoscopic retrograde cholangiopancreatogram

for patients with MRCP evidence of pancreas divisum in whom therapeutic intervention is planned. (See 'Diagnostic evaluation' above.)

- A number of tests have been used in patients with pancreas divisum to determine if they have pathologic narrowing of the minor papilla that may be predisposing them to develop pancreatic symptoms. However, the sensitivity and specificity of these tests are largely undefined and correlation with a response to therapy has not been consistently demonstrated. (See 'Additional tests for pathologic papillary narrowing' above.)

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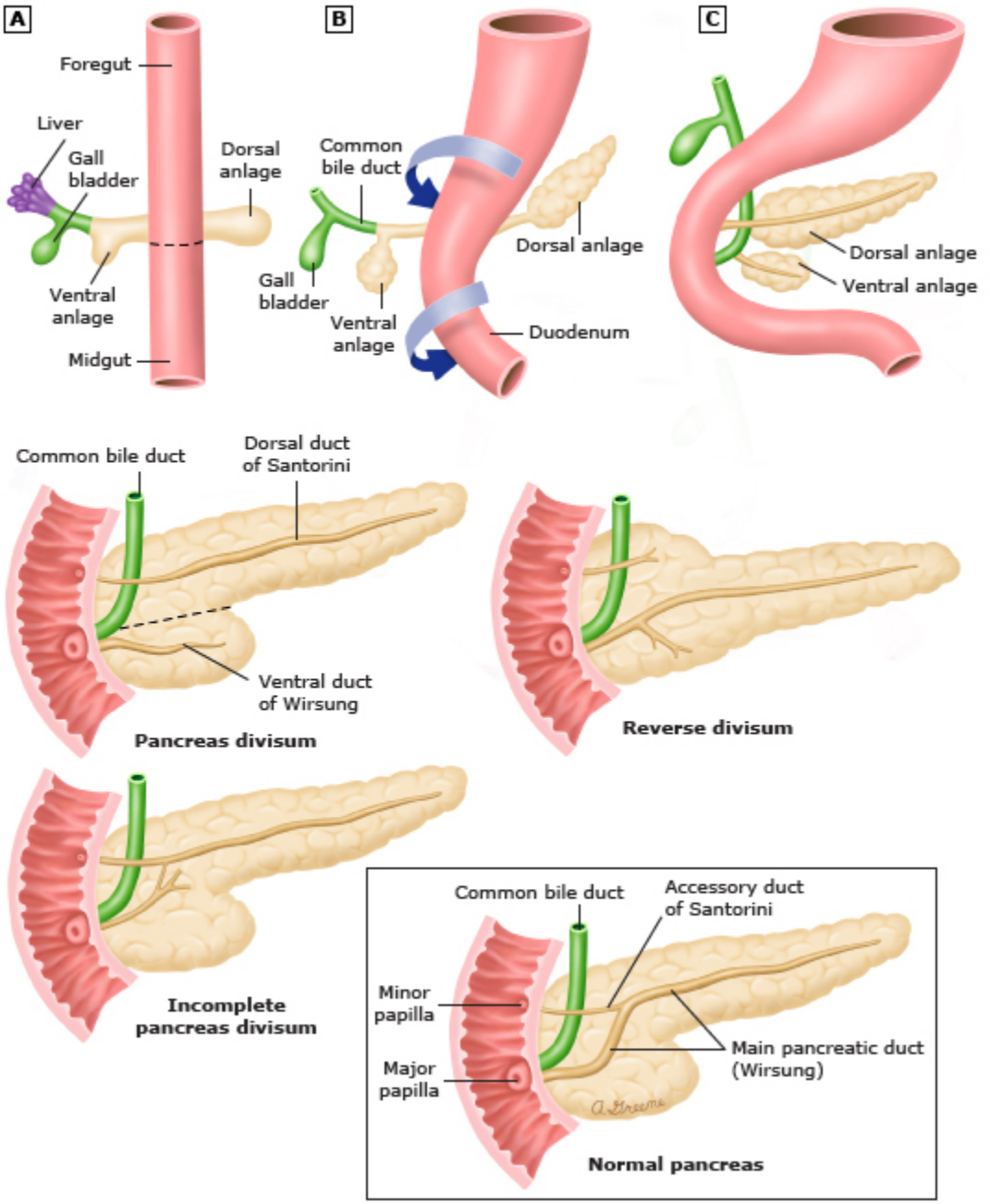
GRAPHICS

Anatomy and embryology of pancreas divisum



The pancreas develops from 2 parts whose ducts are in continuity with the common bile duct. One part is ventral and the other dorsal to the intestinal tract before rotation. The rotation brings the 2 parts together with separate ducts. The duct of the dorsal (larger) part later becomes continuous and enters that of the ventral part, which enters the duodenum with the common bile duct. However, in a congenital malformation (pancreas divisum), the other 2 parts of the pancreas remain distinct, each with its own duct.

Graphic 78995 Version 5.0

Anatomic categorization of congenital pancreatic anomalies and variants

Ventral/dorsal ductal malfusion
1. Pancreas divisum
2. Incomplete pancreas divisum
3. Isolated dorsal segment
Rotation or migration problems
1. Annular pancreas
2. Ectopic pancreas
3. Ectopic papillae
Agenesis or hypoplasia
Ductal duplication
Atypical ductal configuration
Anomalous pancreatobiliary ductal junction
Cystic malformations

Graphic 52761 Version 1.0

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